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## Implications for behavior genetics research: No shared environment left?

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As Plomin & Bergeman (P&B) themselves state repeatedly, an environmental measure is just another phenotype whose genetic architecture does not change because we call it an environmental measure. The exact amount of genetic determination of such measures is an empirical issue and is interesting, as is the heritability of different environmental variables. Equally interesting, however, is the question of what the implications are for traditional behavior genetic models, such as those used in the analysis of twin and sibling data, if environmental indices are genetically influenced. More specifically, what are the consequences if some environmental measures that are perfectly correlated within siblings or twins show an association with the trait under study? A measure that is perfectly correlated within siblings – the number of books in the house, for example – increases the environmental variance shared between siblings. Does the finding that variance in many traditional environmental measures contains a genetic contribution, as P&B illustrate in their target article, or the finding that correlations between measures of the home environment and IQ can be mediated genetically (e.g., Coon et al. 1990), imply that shared environmental variance also is not completely environmental?

In our Figure 1, the elementary model introduced by Plomin, Loehlin & DeFries (1985) to study relations between an environmental index, parental genotype, and offspring's phenotype is extended to families with two siblings. H is the measured home environment (e.g., number of books) that influences the environment ($E$) in which children grow up. Children's environment and genotype ($G$) influence their phenotype (P), a measure such as IQ. As shown by Plomin et al., the correlation between H and P equals ef + rh. Following path analytic rules, we can derive the correlation between the phenotypes of the first and second child as: $ef(ef + rh) + 0.5h^2$. The first part of this expression is the resemblance of siblings induced by the shared environmental variable H. If the trait under study is heritable and parental genotypes are correlated with H, then what emerges as common environmental influences in the children (as, for example, in an analysis comparing identical and fraternal twins) turns out to also include a genetic component!

In an earlier *BBS* target article Plomin & Daniels (1987) pointed out that very little shared environmental influence is usually found for measures of personality and psychopathology. The only trait showing some evidence for common environmental influences is cognition early in life. This is also the measure that correlates reasonably high with some indices of the home environment. Does the fact that H can be genetically influenced mean that there is even less room for shared environment than we already thought?

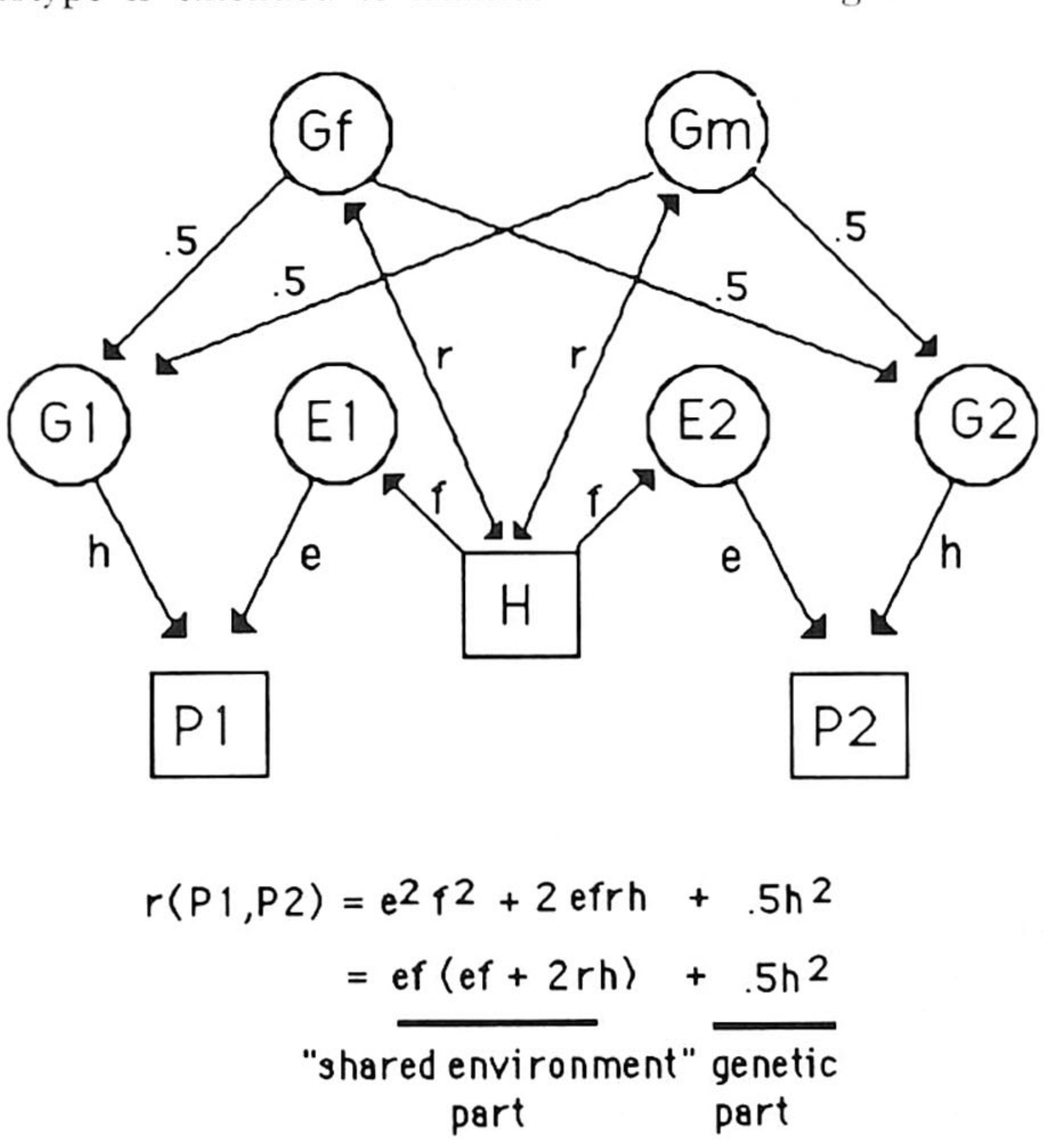


Figure 1 (Boomsma & Molenaar). Path model of the effects on sibling resemblance of a correlation (r) between parental genotypes (G-father and G-mother) and an index of home environment (H). P, E, and G are children's phenotype, environment, and genotype (variables in squares are measured, variables in circles represent unmeasured factors). Path coefficients h, e, and f assess the direct effects of one variable on another. For simplification, the model leaves out direct parental influences on the child's phenotype, assortative mating in the parents, and shared environment in the children that is independent of H.

## Like images refracted: A view from the interactionist perspective

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Plomin & Bergeman (P&B) execute a bold new variation on standard quantitative genetic analyses by treating environmental measures as phenotypes. As devoted gardeners of the interactionist position in the nature/nurture plot, we find many things to admire in this approach, yet other things about which to be wary. Interactionists believe the artificial separation of genetic and environmental influences belies the operation of genetic and environmental action in human development. The evidence presented in this target article helps lay to rest the myth of an independent set of environmental forces.

What is disappointing in the discussion by P&B is their failure to adopt a clear interactionist position. Repeated use of the term "genetic influence" clouds the contribution of an interesting new approach to the nature/nurture issue. Based on information developed using quantitative genetic analyses, the term "influence" simply refers to the increased probability of some score on an environmental measure, not a genetic blueprint (Oyama 1985). Neither can one leap from population statistics to any particular "mechanism" that drives the course of individual development. As surely as environmental measures do not actually represent some set of environmental forces "out there" in reality, neither do those measures reveal a specific set of genetic directions "in there." P&B essentially concede this point, yet they continue to use the term genetic influence; they insist that they are not really talking about environments as if they were phenotypes, but about *measures of the environment* as phenotypes. There may be a bit too much cleverness in this position. It isn't always easy to keep such distinctions separate, especially when one is trying to derive practical implications from the results. Johnston (1987) presents a revealing account of how myths about genetic or environmental influence persist despite the overwhelming evidence in favor of the interactionist position.

It is all too easy to forget that methods in science are simply metaphors in action. As we begin to layer these metaphors one